



Extracranial and Transcranial Ultrasound Assessment in Patients with Suspected Positional ‘Vertebrobasilar Ischaemia’

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Abstract *Objectives:* A diagnosis of ‘positional’ vertebrobasilar ischaemia is considered in patients presenting with dizziness/vertigo during lateral neck rotation/extension and is attributed to bony ‘nipping’ of the vertebral artery (VA). This study reviewed our experience with extracranial and transcranial ultrasound to determine whether a diagnosis of ‘positional’ vertebrobasilar ischaemia was associated with any changes in flow in the extracranial VA and the P1 segment of the posterior cerebral artery (PCA) during head turning.

Methods: A retrospective case note review was undertaken in 46 patients with an accessible window for transcranial Doppler who had undergone extracranial and transcranial assessment of flow velocity and flow directionality in the VA and PCA while the head was moved into positions that normally triggered the patient’s symptoms.

Results: Positional ‘vertebrobasilar symptoms’ were triggered by lateral head rotation in 35 patients (76%), while 11 (24%) developed symptoms following neck extension. Only one patient was found to have a significant carotid stenosis (symptoms unchanged following carotid endarterectomy) and none had significant disease in the extracranial VAs. None of the patients exhibited any change in extracranial VA flow during head turning/extension and none had reversal of flow either. Similarly, there was no change observed in the PCA flow characteristics during head turning. The majority of patients (74%) were subsequently referred to the Ear, Nose and Throat (ENT) department, and 94% of the patients noted an improvement in symptoms following entry into a vestibular rehabilitation programme.

Conclusions: A diagnosis of ‘positional’ vertebrobasilar ischaemia should be made with extreme caution and only after a specialist assessment in a Balance Centre.

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The vertebral arteries (VAs) arise from the subclavian arteries and pass posterosuperiorly between longus colli and scalenus anterior before passing superiorly through the foramen in the transverse process of the sixth cervical vertebra. The VAs then ascend via the transverse foramina of the upper cervical vertebrae to the level of C2, where they pass posterolaterally around the posterior arch of C1 before passing between the atlas and occiput. Having pierced the dura at the level of the foramen magnum, the VAs meet at the level of the medulla/pons to become the basilar artery (BA). There are, therefore, a number of points where the VA can be compressed by bony/soft-tissue structures, either as a consequence of an anatomical anomaly or because of a degenerative change.

It has become almost conventional to consider a diagnosis of 'positional' vertebrobasilar ischaemia in patients who present with dizziness/vertigo or faintness where these symptoms are specifically associated with either lateral neck rotation or extension. It is generally assumed that these patients have 'nipping' of the extracranial VAs sufficient to cause haemodynamical compromise and the onset of posterior fossa symptoms. Not surprisingly, patients may be labelled as having suffered vertebrobasilar transient ischaemic attacks (TIAs), often without further investigation.

Technological developments in ultrasound imaging now mean that it is possible to insonate and image the extracranial and basal intracranial circulation to ascertain whether these patients display any change in flow patterns in the VA/posterior cerebral artery (PCA) vessels during head rotation and extension. A failure to elicit any change in flow haemodynamics would render any diagnosis of positional 'vertebrobasilar ischaemia' extremely unlikely.

Materials and methods

Since 2000, a standardised ultrasound protocol has been employed in patients referred to the Vascular Unit with suspected positional vertebrobasilar ischaemia and a retrospective audit was undertaken of the findings in 46 patients

presenting between January 2000 and July 2007 (and who had an accessible temporal window for transcranial Doppler ultrasound). Each had been referred with a diagnosis of suspected positional vertebrobasilar ischaemia. The Leicestershire, Northamptonshire and Rutland Research Ethics Committee advised that this study did not fall under the remit of the NHS Research Ethics Committee as it was an audit/service evaluation.

Extracranial Duplex examination of the carotid and vertebral arteries

This was performed using a Philips HDI 5000 Duplex scanner with a 7–4 MHz linear array transducer, with the patient lying in a supine position in a darkened, quiet room. The extracranial carotid arteries were assessed in order to exclude any haemodynamically significant lesion (>70%), while also excluding abnormal low-velocity or high-resistance flow that may indicate a dissection or distal siphon disease. The proximal VAs were then imaged, as were the intervertebral segments of the extracranial VAs and assessed for the presence of stenosis >50%, tortuosity, patency, direction of flow, asymmetry of size or flow characteristics, such as high-resistance flow or increased systolic rise time.

The patient was then placed in a sitting position with the head in a neutral position, looking directly ahead. The extracranial VAs were re-imaged and the best intervertebral segment of this vessel selected for imaging (always above C6). The flow patterns were monitored during a variety of head movements (Fig. 1A) starting with the right VA. The head was first rotated to the left and then to the right. Second, the head was extended and then rotated to the left/right. Third, the head was maximally flexed and then rotated from left to right. Finally, the patient was asked to position his/her head in any other position that normally triggered the symptoms. A similar protocol was then carried out on the left VA. Any change in flow velocity or directionality in the intervertebral segment of the VA was documented (Fig. 1B).

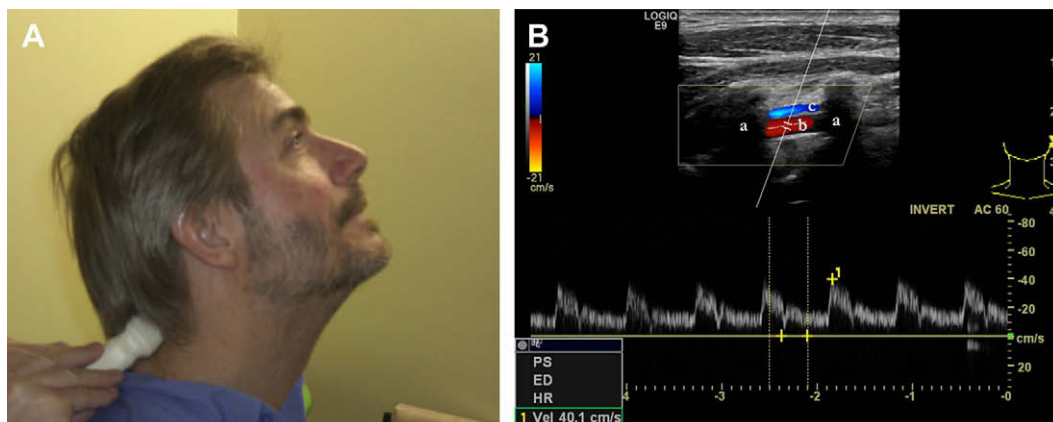


Figure 1 (A) Insonation of an intervertebral segment of the right VA while the head is extended using colour Duplex ultrasound. (B) Flow velocity and directionality in an intervertebral section of the VA is monitored at baseline and then when the head is moved into extension, rotation, etc. Key: (a) acoustic shadow caused by transverse processes of cervical vertebrae, (b) vertebral artery, (c) vertebral vein. The cursor is placed over the intervertebral segment of the VA to obtain a waveform.

Intracranial transcranial Doppler examination

If there was a good transcranial window, the basal circulation was imaged using a Philips HDI 5000 scanner with a 4–2 MHz phased-array transducer (Fig. 2A). Using a phased-array transducer via the transtemporal window, the middle cerebral arteries (MCAs) and PCAs were identified and imaged using colour flow (Fig. 2B). The P1 segment of the PCA normally has flow towards the transducer, whilst the P2 segment will normally demonstrate flow in the opposite direction as the artery curves away from the probe. Note that in Fig. 2A, it is often possible to image most of the circle of Willis in one plane. Spectral Doppler recordings were taken from the P1 segment of the PCA at a typical depth of 6–7 cm (Fig. 2B). If, however, the transcranial window for colour Duplex imaging was poor, the MCA and PCA vessels were insonated using a Scimed TC22 2 MHz pulsed, transcranial Doppler (Fishponds, Bristol, UK).

With the patient in a seated position looking directly ahead and using the transtemporal window, the P1 segment of the PCA is located by first insonating the MCA at a sample depth of 5.5 cm. The sample depth is then increased to 6 cm and the transducer gently angled posteriorly in order to intercept the PCA. Flow direction in the P1 segment should be towards the transducer. The sample depth can be increased to 7 cm if required to detect the PCA. The Doppler waveform is assessed and any inter-hemispheric abnormality noted (such as reduction in flow velocity, damping or evidence of high-resistance flow). Flow in the P1 segment of the PCA is then monitored on both sides of the brain during the following head manoeuvres (Fig. 2A): head extension, head rotated to the left, head extended and rotated to the left, head flexed and rotated to the left, head extended and rotated to the right and head flexed and rotated to the right. Finally, the patient was asked to position his/her head in any other position that normally triggered the symptoms. The suboccipital window (head flexed with chin on the chest) opens up the space between the cranium and the atlas vertebra. However, this only allows very limited access for

imaging during head-extension and head-turning manoeuvres. It is not possible to reliably insonate the intracranial VA and then maintain the probe position and vessel insonation while the head is being moved.

Results

Forty-six patients (males = 16, females = 30) with an accessible transcranial window were referred to the Vascular Unit at Leicester Royal Infirmary with a suspected diagnosis of positional vertebrobasilar ischaemia. The mean age was 69 years (range: 32–98 years). The majority ($n = 40$) of patients complained of dizziness/vertigo when their heads were rotated laterally or following neck extension, five felt faint with neck movement, two developed nausea while one became ataxic. Only six (13%) had a prior history of cervical neck pain. Positional vertebrobasilar symptoms were triggered by lateral head rotation in 35 patients (76%), while 11 (24%) developed symptoms following neck extension.

Only one patient (2%) was found to have a significant carotid stenosis (75%). There had been no preceding carotid territory symptoms. A carotid endarterectomy was performed in this patient following completion of investigations, but this made no difference to the patient's positional symptoms. No significant extracranial VA stenoses were detected in any patient, although it was not unusual for one VA to be of a smaller calibre than the other. More importantly, there was no change in VA flow (velocity or flow directionality) when the head was either extended or laterally rotated in any patient. Similarly, flow in the P1 segment of the PCA showed normal antegrade flow, with no changes in flow velocity or directionality when the head was placed into positions that normally triggered the patient's symptoms. Thus, each patient had paired extracranial and intracranial studies and none showed any changes in flow velocity or directionality.

Thirty-four patients (74%) were subsequently referred to the Balance Centre for formal assessment, where the

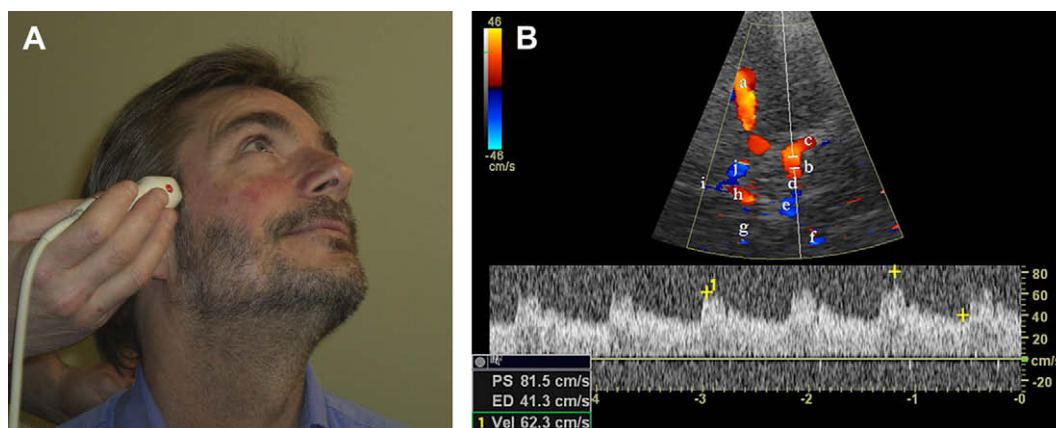


Figure 2 (A) Insonation of the P1 segment of the right PCA artery while the head is extended and rotated to the left. (B) Colour transcranial Duplex image of the principal components of the circle of Willis. Key: (a) = right middle cerebral artery mainstem, (b) P1 segment of the right PCA, (c) P2 segment of the right PCA, (d) confluence of the basilar artery, (e) P1 segment of the left PCA, (f) P2 segment of the left PCA, (g) left middle cerebral artery, (h) A1 segment of the left anterior communicating artery, (i) A2 segment of one of the anterior cerebral arteries and (j) A1 segment of the right anterior cerebral artery. The cursor is located over the P1 segment of the right PCA to obtain a waveform for analysis.

majority of patients were subsequently diagnosed as having benign positional vertigo and entered into a vestibular rehabilitation programme. Overall, 94% of patients referred to the Balance Centre improved following therapy.

Discussion

Cadaveric blood-flow studies were among the first to demonstrate that neck rotation could cause compression of the vertebral arteries at the C1–C2 and C5–C6 levels.¹ Since then, it has become almost conventional to consider a diagnosis of 'positional' vertebrobasilar ischaemia in any patient who presents with dizziness/vertigo or faintness where these symptoms are specifically associated with either lateral neck rotation or extension. Once such a history has been elicited, few physiotherapists will proceed with rotational cervical spine manual therapy until investigations have excluded a vascular cause.

It is generally assumed that patients with positional vertebrobasilar ischaemia have 'nipping' of the extracranial vertebral arteries sufficient to cause haemodynamic compromise causing the onset of posterior-fossa-type symptoms. Not surprisingly, many may then be erroneously labelled as having suffered true vertebrobasilar TIAs. A recent systematic review of the literature has highlighted the conflicting haemodynamic data regarding this subject.² Out of the 20 published studies, seven reported no changes in VA or PCA blood flow, while 13 described varying changes in blood flow (reversal, complete occlusion and reduced flow). One of the main problems, however, is that there is no consensus definition for this condition, most published trials involve a very heterogeneous group of patients with wide age ranges and many have differing durations of symptoms.

While the current study found no examples of any changes in flow velocity or directionality in any of our patients, others have documented complete cessation of blood flow in the PCA with head extension and rotation that also precipitated the patient's symptoms (Fig. 3).³ This case clearly confirms that the condition really does exist; however, our findings suggest that it is extremely rare. The fact that virtually all of the patients in this series who were subsequently referred to the Balance Centre for further investigation and management improved following implementation of a vestibular rehabilitation programme suggests that none of our patients had a vascular cause for their symptoms.

Our study does, of course, have limitations. First, we did not have any age-matched controls for comparison. Second, we did not routinely assess flow in the carotid arteries during neck rotation. We had not previously considered this necessary, but others have reported changes during provocative head movements.² Third, although several patients reported symptoms during extracranial and intracranial ultrasound assessment, we did not routinely document when these occurred. These would have been valuable data to evaluate. Finally, the VAs can be difficult to examine throughout their length, and it is

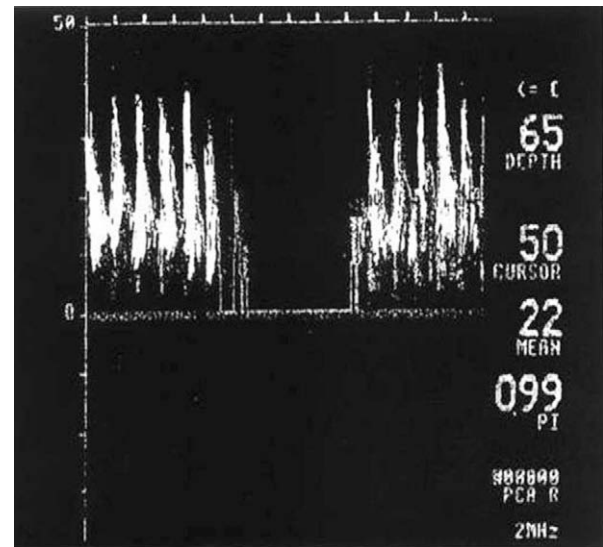


Figure 3 Cessation of flow in the posterior cerebral artery during a combination of neck extension and flexion. Flow was immediately restored once the neck returned to the neutral position. The patient developed vertebrobasilar symptoms during the period of flow cessation. Reproduced with permission by Elsevier from Terenzi, Transcranial sonography and vertebrobasilar insufficiency. *J Manipulative Physiological Ther* 2002;25:180–3.

possible that VA occlusion did develop in some patients, which was either unnoticed or compensated for by an excellent collateral circulation. In Terenzi's case (reproduced in Fig. 3), the patient had an anomalous circle of Willis, with the right VA giving rise to the basilar artery. The left VA terminated as the posterior inferior cerebellar artery.³ It may be, therefore, that VA compression is more likely to give rise to symptoms if there is a co-existing anomaly of the circle of Willis.

In conclusion, the results of this study suggest that extracranial compression of the VAs with head extension or rotation is an extremely rare cause of positional vertebrobasilar ischaemia and most often will probably have inner-ear pathology. As a consequence, this diagnosis should never be made without first having sought a specialist referral to the Balance Centre.

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